THE ŒSOPHAGEAL LEAD

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Although electrocardiograms were taken from the œsophagus as long ago as 1906 (by Cremer) it was Brown (1936) who first showed the possibilities of the method and made the lead of clinical significance. The œsophagus is in close relation to the left auricle for about 5 cm. below the level of the fifth dorsal vertebra; lower down it is in relationship to the right auricle and the diaphragmatic surface of the ventricles. Thus the œsophageal electrocardiogram can be divided into three sectors. The supra-auricular complexes resemble those in lead VR with negative P waves. At the auricular level P waves are replaced by intrinsicoid deflections which are usually much greater in amplitude than are found in any other lead. At the ventricular level P is positive and the ventricular complexes are like those in lead VF. Between the auricular and ventricular levels there is a transitional zone of varying depth, in which the P waves are becoming peaked, Q waves appear, and T may be negative.

Method. An electrode was constructed from a Ryle's duodenal tube, as described by Butterworth and Poindexter (1946), with a tip of German silver substituted for the rubber end. Wires running up the bore of the tube connected the German silver electrode to a terminal at the proximal end of the tube. To this was attached the left-arm terminal from the galvanometer, the indifferent electrode being the V central terminal of Wilson.

The tube was either swallowed or passed through the nose; most patients have found it easier to swallow it. In the earlier cases the tube was passed down to about 46 cm. from the teeth. Later 56 cm. was found to be more satisfactory since at this level the tip will have reached the stomach in patients of every build. Records were taken at 2-cm. intervals as the tube was withdrawn until a level of about 26 cm. was reached. No attempt was made to check the position of the electrode by means of fluoroscopy. The œsophageal electrocardiogram can be calibrated by means of the auricular intrinsic deflections, which are always to be seen at some level except in cases of auricular fibrillation. With that exception, unless they are found in some of the strips, it can be assumed that the tube has coiled in the throat. All the strips will then be similar and will resemble lead VR. The correct passage of the electrode can therefore be judged by a scrutiny of the curves, and the record can thus be taken by a cardiographer.

Swallowing a metal end-piece is not easy for orthopnœic patients and several were unable to do so. In two instances it was found that the tube had coiled in the throat.

Seventy records were obtained on 65 patients. At the outset patients with healed or doubtful posterior infarcts were examined, and those with different types of auricular arrhythmias. Patients with bundle branch block were later included in order to see which ventricle was mainly responsible for the deflections on the diaphragmatic surface of the heart.

The Normal Œsophageal Curve

Auricular Deflections. At the lowest levels P is upright. As the tube is withdrawn it becomes more peaked and narrow until the sharp auricular R appears, the descending limb of which is the intrinsic deflection (Fig. 1). The level at which the change occurs varies considerably with patients

of different builds and with different positions of the heart (Oblath and Karpman, 1951). The lowest level at which an intrinsic deflection was observed was 52 cm. from the mouth; the highest was 32 cm. The auricular R may be notched. At upper auricular levels the descending limb is prolonged into an S wave and R disappears. Leads taken from the right auricular cavity have shown that negative deflections occur at the upper auricular levels, when the electrode is near the sinus node, and diphasic deflections at the mid-auricular zone (Schlesinger *et al.*, 1949). The longest stretch over which intrinsic deflections were obtained in any patient was 22 cm. In some cases they only appeared at one level. The size of the deflection varied greatly but in half of the records it was 6 mm. or more at the most favourable level. The tallest auricular R recorded was 10 mm., the deepest S was 13 mm., giving a total of 23 mm. for the intrinsic deflection. This compares with 10 and 10 mm. found by Oblath and Karpman (1951), and 25 mm. for the total intrinsic deflection noted by Enselberg (1951). The auricular QRS has a duration of about 0·06 sec. The auricular T wave is seen best in cases of complete heart block since, when the rhythm is normal, it coincides with the ventricular deflections. It is a broad shallow deflection which is opposite in sign to the major initial deflection (Fig. 2).

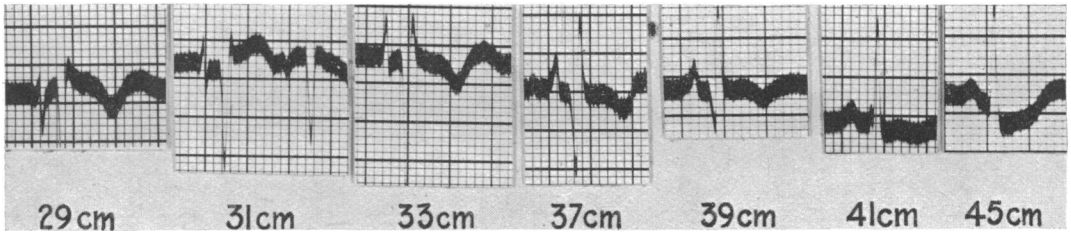


FIG. 1.—*Normal œsophageal curve.* Auricular intrinsicoid deflection, predominantly negative at 29 cm., becomes positive at 33 cm. P waves are peaked at 39 cm. and normal at 41 cm. An auricular premature systole at 31 cm. Ventricular complexes have a deep Q, small R, and negative T (left ventricular cavity potentials) at 33 cm. Q disappears at 41 cm. when P becomes normal. RS–T junction is depressed at 45 cm.

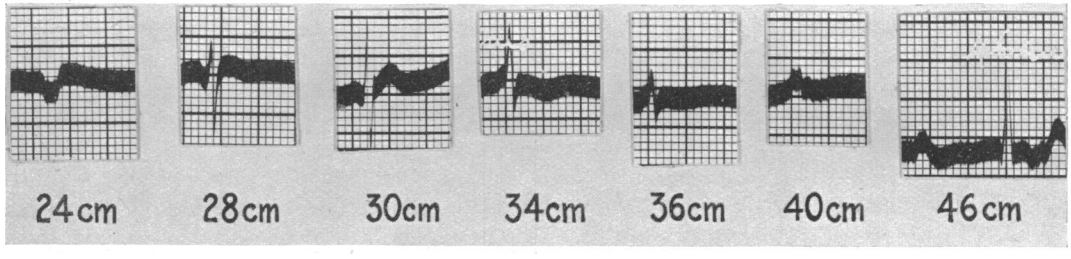


FIG. 2.—*Complete heart block showing auricular deflections.* The auricular T wave is seen as a blunt upward deflection at 28 cm. when the intrinsicoid deflection is mainly negative, and as a broad shallow deflection at 34 cm. when it is positive. The auricular Q–T interval is 0·36 sec.

The normal ventricular deflection at the lowest levels consists of an R which may be preceded by a small Q (Sandberg *et al.*, 1950). A shallow S frequently follows, but, apart from left bundle branch block, a deep S occurred only in five horizontal hearts. In this position the œsophagus faces the right ventricle (Myers and Klein, 1948). T is upright but there may be depression in health of the S–T junction (Fig. 1 and 4). As the P wave becomes peaked, the ventricular Q deepens or appears for the first time. In 4 cases the ventricular Q preceded the P wave changes by 4 cm. and in 7 more by 2 cm. but usually they were simultaneous. At the same time the T wave flattens and then becomes negative. At the auricular levels the ventricular deflections consist of a large Q and a small R with a negative T and they represent the potentials of the cavity of the left ventricle, since the œsophagus is in close relation to the left auricle at this point (Sandberg *et al.*, 1950*b*).

At supra-auricular levels the ventricular deflections, while retaining in general this form, diminish in size, and come to resemble those of lead VR.

The Œsophageal Lead in Arrhythmias

Heart Block. In some cases of complete heart block the P waves are small and cannot easily be made out. In such cases the œsophageal lead enables the independent auricular rhythm to be defined with certainty (Fig. 3).

Auricular Premature Contractions are clearly seen (Fig. 4) and their point of origin in terms of

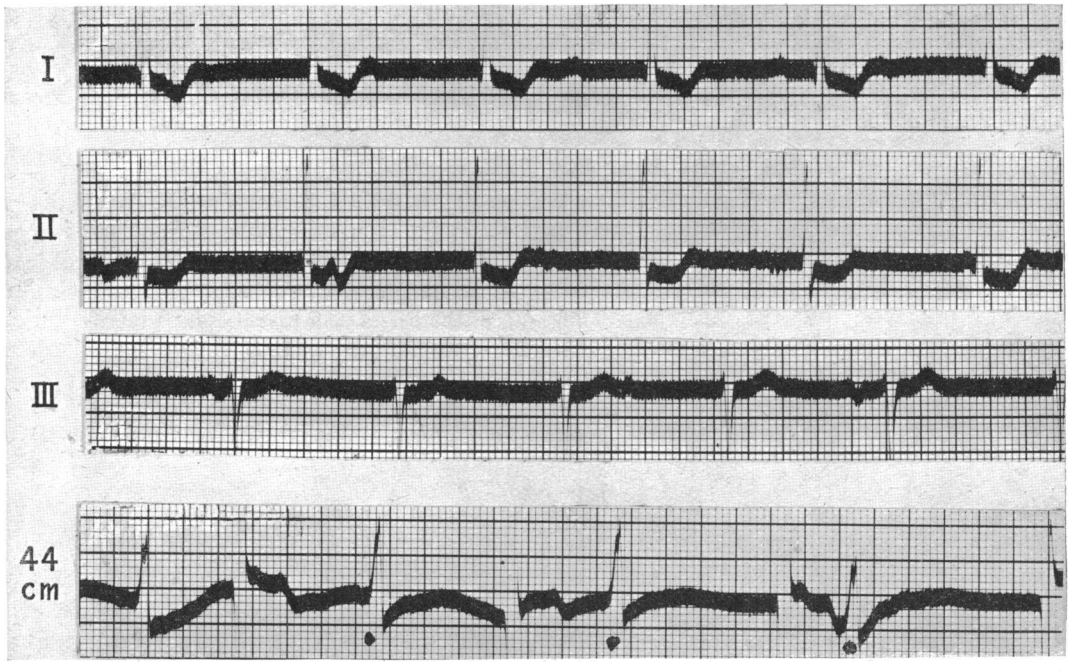


FIG. 3.—*Complete A-V dissociation.* P waves cannot be made out with certainty in the standard leads. Notched auricular waves are obvious in the œsophageal lead at 44 cm. (lowest curve). Auricular rate, 45; ventricular rate in standard leads, 60; in œsophageal, 40.

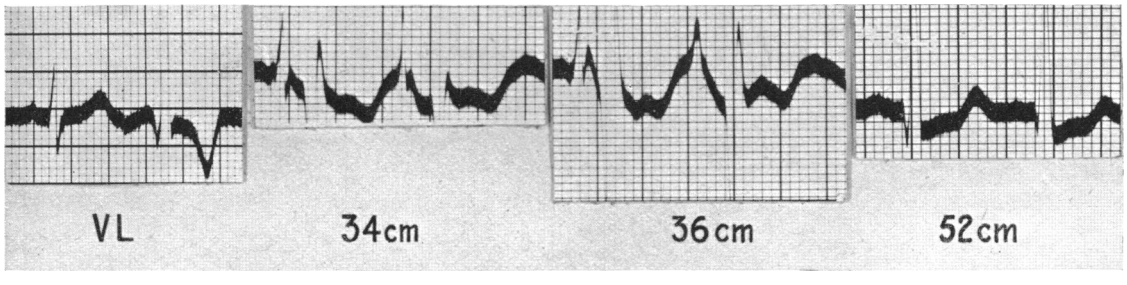


FIG. 4.—*Premature auricular systoles with ventricular aberration.* Lead VL shows a premature systole with marked aberration. P is not well defined. At 34 cm. the normal auricular deflection has an S wave which is absent in the premature beat. At 36 cm. the normal beat has an auricular R wave, the premature beat a peaked P wave. The premature beat is therefore arising from a higher level than the normal probably in the left auricular appendage. Note also the moderate ventricular Q and depressed RS–T junction in the normal complex at 52 cm.

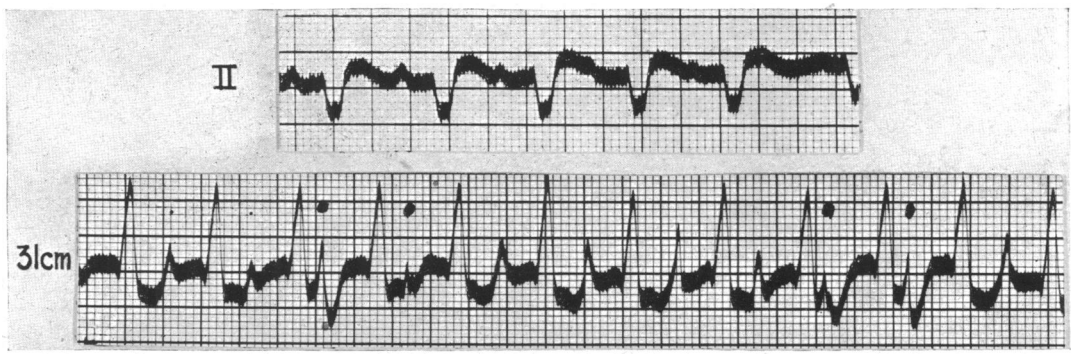


FIG. 5.—*Multiple auricular premature systoles.* Case 25, left bundle branch block (see also Fig. 13). Premature systoles not well seen in lead II. At 31 cm. they occur just after the ventricular QRS at the 3rd, 4th, 9th, and 10th beats with a P–R interval of 0·28 sec.

distance from the sino-auricular node can be made out. In Fig. 4 they are probably arising from the left auricular appendage. Multiple premature auricular systoles may be difficult to distinguish in the conventional leads (Fig. 5). The early large P waves are obvious in the œsophageal lead.

Auricular Tachycardia. In some cases of auricular tachycardia standard leads may simulate auricular fibrillation, but the œsophageal lead at auricular levels reveals the regular auricular deflections (Fig. 6 and 7). It seems likely that the œsophageal lead will show that all cases of repetitive tachycardia are due to an ectopic focus in the auricles.

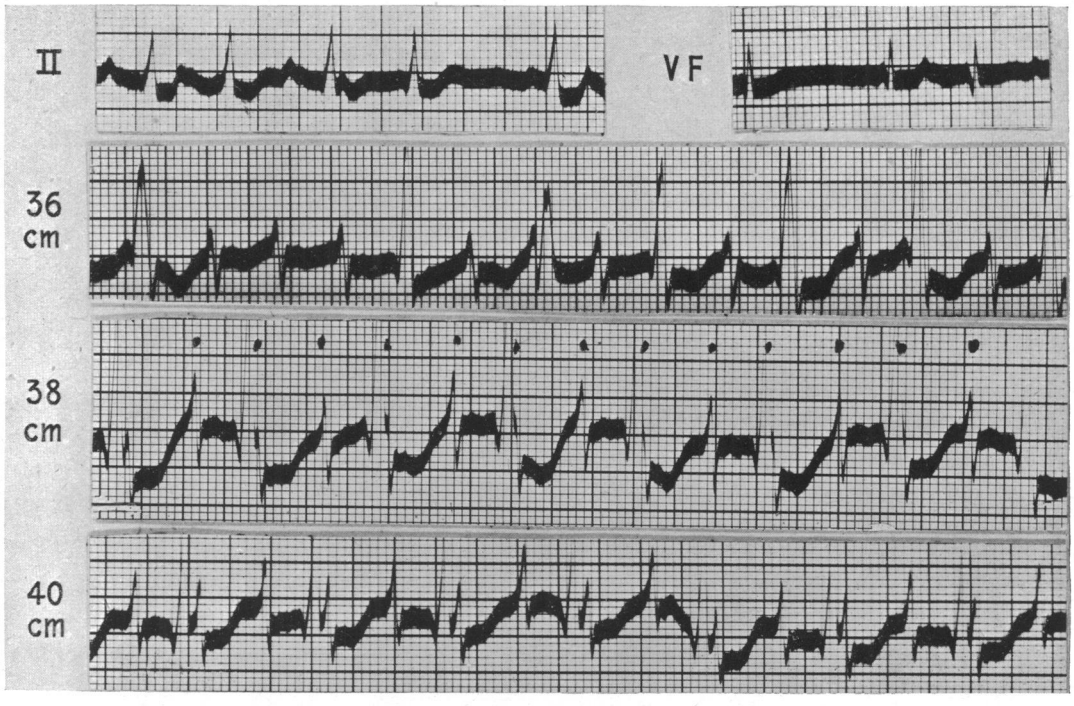


FIG. 6.—*Auricular Tachycardia with A-V heart block.* Leads II and F simulate auricular fibrillation. Œsophageal leads show an auricular tachycardia at 190. At 36 cm. there is a mixed 4 to 1 and 2 to 1 block: at 38 and 40 cm. the block is 2 to 1.

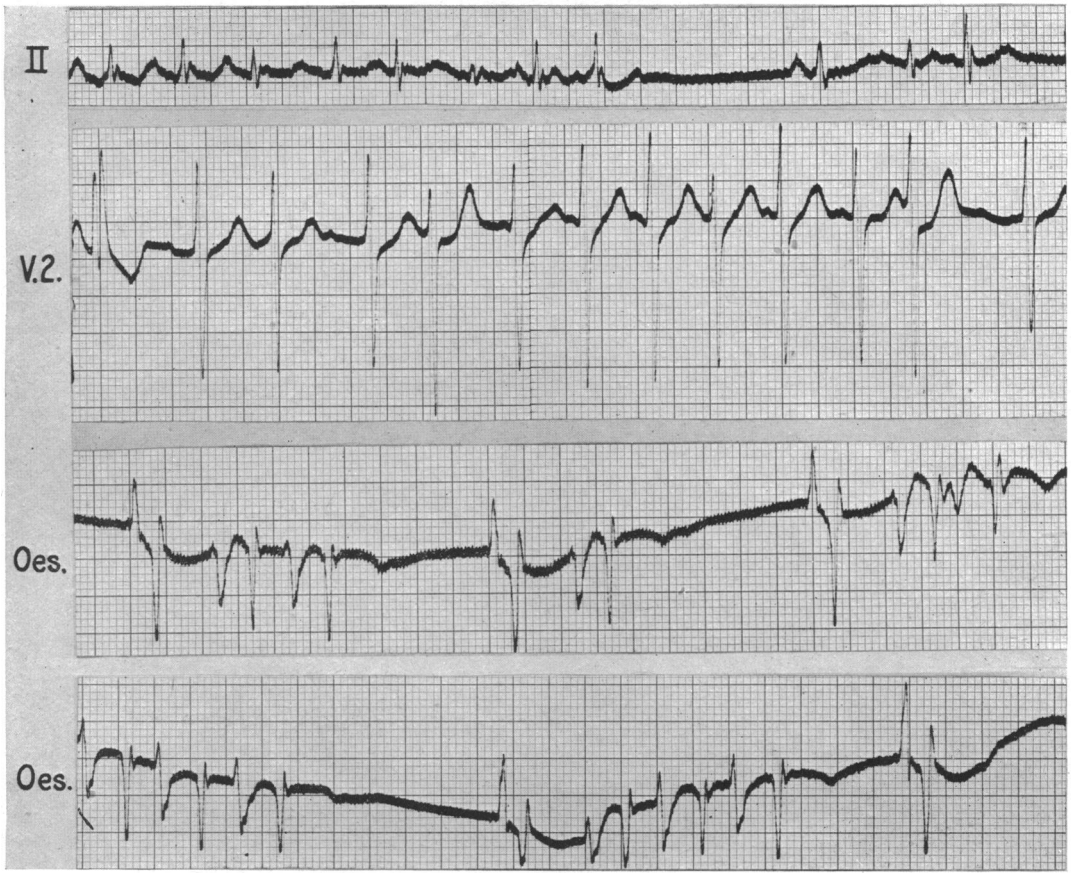


FIG. 7.—*Repetitive Auricular Tachycardia.* Attacks present for 40 years. Lead II shows two short attacks with a normal complex between them. Lead V2 shows a longer attack. All attacks simulate auricular fibrillation. The œsophageal lead (upper strip) shows premature auricular systoles arising singly and in couples from a focus low down in the auricle. The auricular complexes are broad and notched. The lower strip shows short paroxysm of auricular tachycardia at a rate of 150.

Auricular Flutter. In the six records obtained, the auricular rate varied from 240 to 360. At auricular levels a sharp auricular intrinsic deflection occurred which was similar to the normal auricular intrinsic deflection or to the deflection obtained in auricular tachycardia. In all the records there were one or more levels where the base line between the intrinsic deflections was flat, although, as noted by Brown (1936) and Enselberg (1951), above and below that level the base line tended to ascend. On the whole the deflections were large, the total intrinsic deflection varying from 11 to 22 mm. At the lower levels they were predominantly negative becoming positive at higher auricular levels, suggesting an abnormal site of origin of the impulse (Fig. 8 and 9).

Auricular Fibrillation. Of 10 cases of auricular fibrillation 9 had fibrillary waves similar to those seen in other leads (Fig. 10). The other record was taken during the phase of fibrillation that followed the treatment of flutter with digitalis before normal rhythm was restored, and in it the auricular deflections were large, reaching 11 mm. But they were also broad, having an auricular QRS of 0·08 sec. duration, and there was never a sharp auricular intrinsic deflection with a return to the base line. Instead, the well known characteristics of total irregularity, and of waxing and waning in each strip, were present. The features were what might be anticipated from a circus with a changing axis constantly casting off impulses as it revolves.

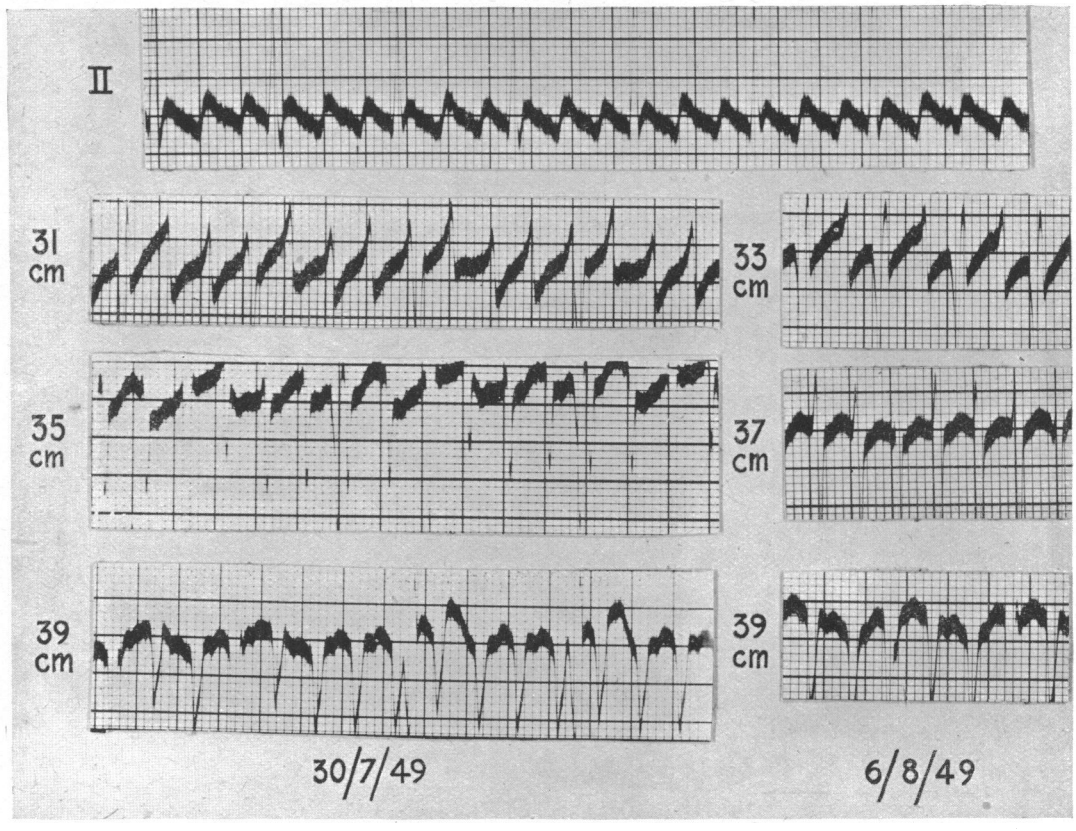


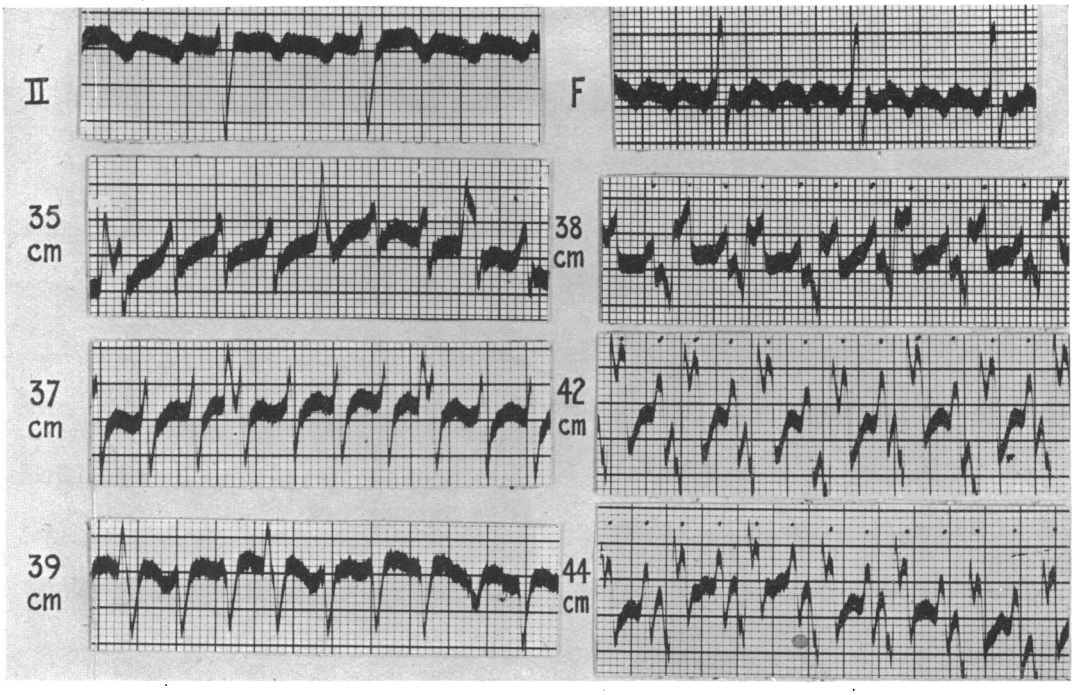
FIG. 8.—*Auricular Flutter.* Case 20. Two records. Flutter rate is 300. At 39 cm. auricular complexes are negative. At 35 and at 37 cm. sharp narrow auricular intrinsic deflections are seen which are predominantly negative. In the second record at 37 cm. the appearances between the intrinsic deflections suggest auricular T waves (see Fig. 2 at 30 cm.). At 31 and 33 cm. the auricular complexes are positive and there is an ascending base line.

Ventricular Tachycardia. Two records were taken during paroxysms of ventricular tachycardia (Fig. 11). In both the independent auricular rhythm was easily seen, since at auricular levels the auricular deflections dwarfed the ventricular.

Ventricular Premature Contractions. At ventricular levels these can be seen well enough, but they are equally obvious in other leads and the œsophageal lead does not offer any advantage.

IN CARDIAC INFARCTS AND BUNDLE BRANCH BLOCK

Posterior Cardiac Infarcts. It was hoped that the œsophageal lead would provide additional information in posterior infarction but the results have proved disappointing. Five patients with recent infarction, 5 with healed infarcts, and 9 with doubtful infarcts were examined. In no case was any information obtained from the œsophageal lead that was not already available in lead VF. Those with signs of recent or healed infarcts in lead VF showed similar signs at the ventricular levels of the œsophageal lead (Fig. 12). In those with only a small Q and a flat T in VF the œsophageal lead was either similar or else it was normal. High posterior infarcts occur too near to the transitional zone to make the œsophageal curves reliable, since the Q waves and negative T waves may form part of a normal transition to cavity potentials. It appears that the left leg lead (VF) reflects the potentials of the diaphragmatic surface of the ventricle as accurately as does the œsophageal (Scherlis



A B

FIG. 9.—*Auricular Flutter.* (A) Case 17. Flutter rate is 240. At 39 cm. auricular complexes are negative. At 37 cm. sharp, narrow intrinsic deflections which are predominantly negative. Auricular T waves seen between intrinsic deflections. At 35 cm. complexes are predominantly positive and there is a tendency towards an ascending base line. (B) Case 51. Flutter rate is 280. At 38 cm. where the auricular complexes are positive the base line is isoelectric.

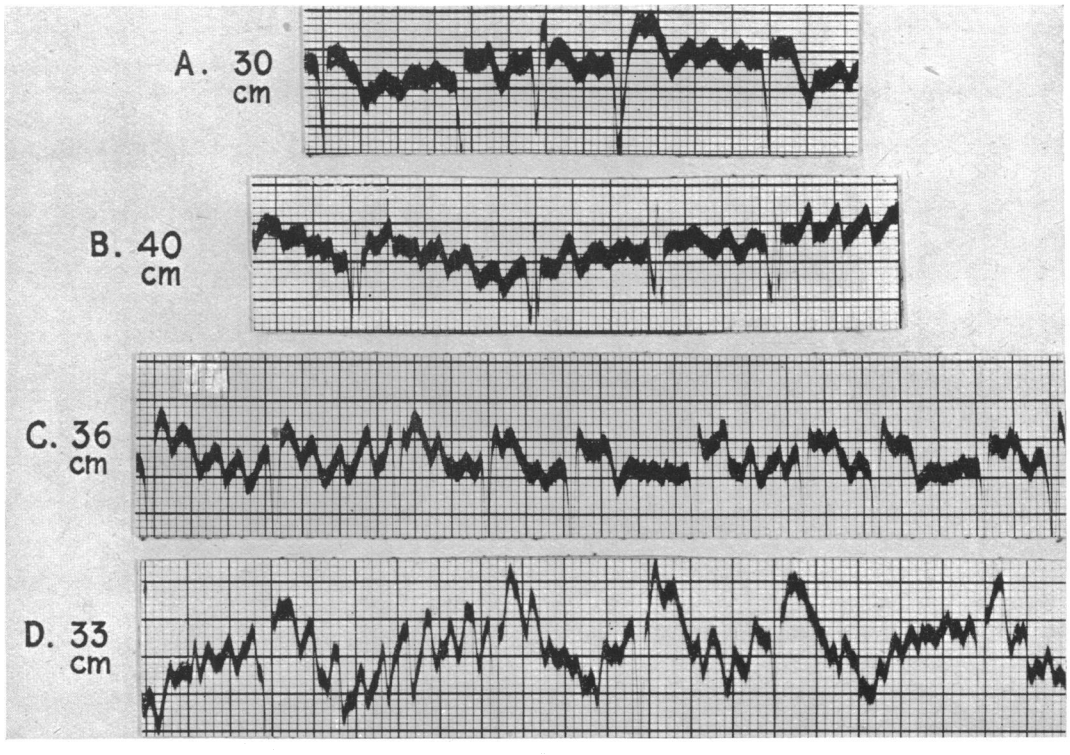
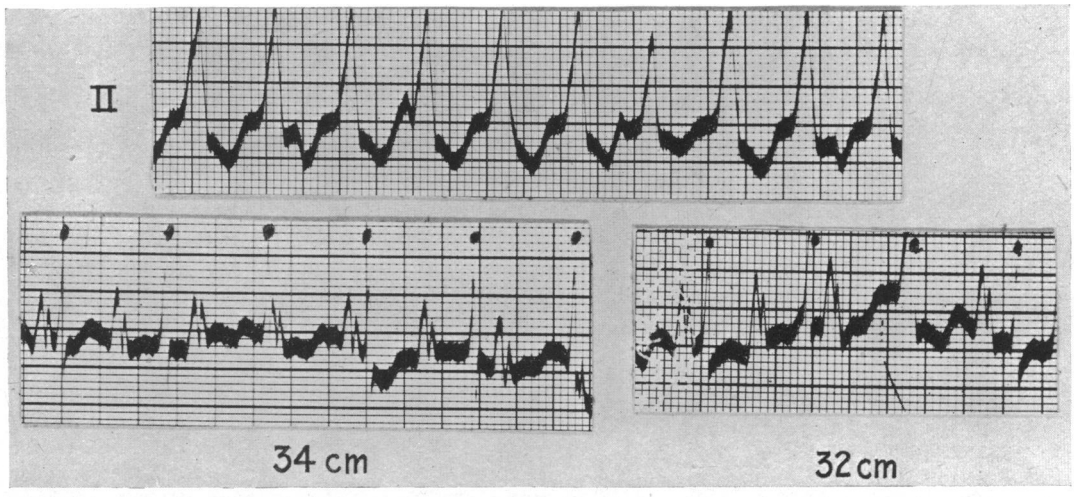


FIG. 10.—*Auricular Fibrillation.* (A), (B), and (C) (Cases 47, 45, 58) show the largest fibrillary waves obtained in patients with established fibrillation. In (D), taken during the treatment of flutter with digitalis, large deflections up to 11 mm. are present at times but they are broad and there is never any return to the base line.



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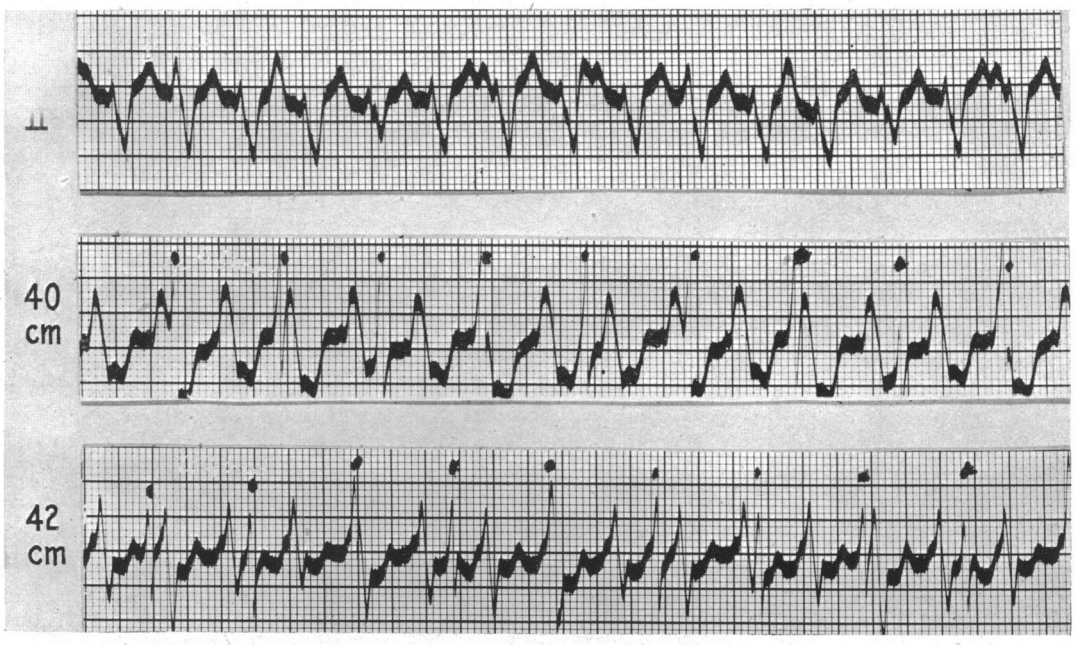

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FIG. 11.—*Ventricular Tachycardia.* (A) Rate 146. An independent auricular rhythm at 110 can just be made out in lead II, but is obvious at 34 cm. where the auricular complexes are larger than the ventricular. (B) Rate 160. Auricular rhythm at 100 clearly seen at 42 cm.

et al., 1951). It also seemed possible that the œsophageal electrode just before and after it enters the stomach might face the right ventricle in some cases and not the left. To test this possibility patients with bundle branch block were examined.

Left Bundle Branch Block. Ten cases were examined. In 5 the deflections of the œsophageal lead at the ventricular level resemble lead V5, showing that the electrode was facing the left ventricle. In

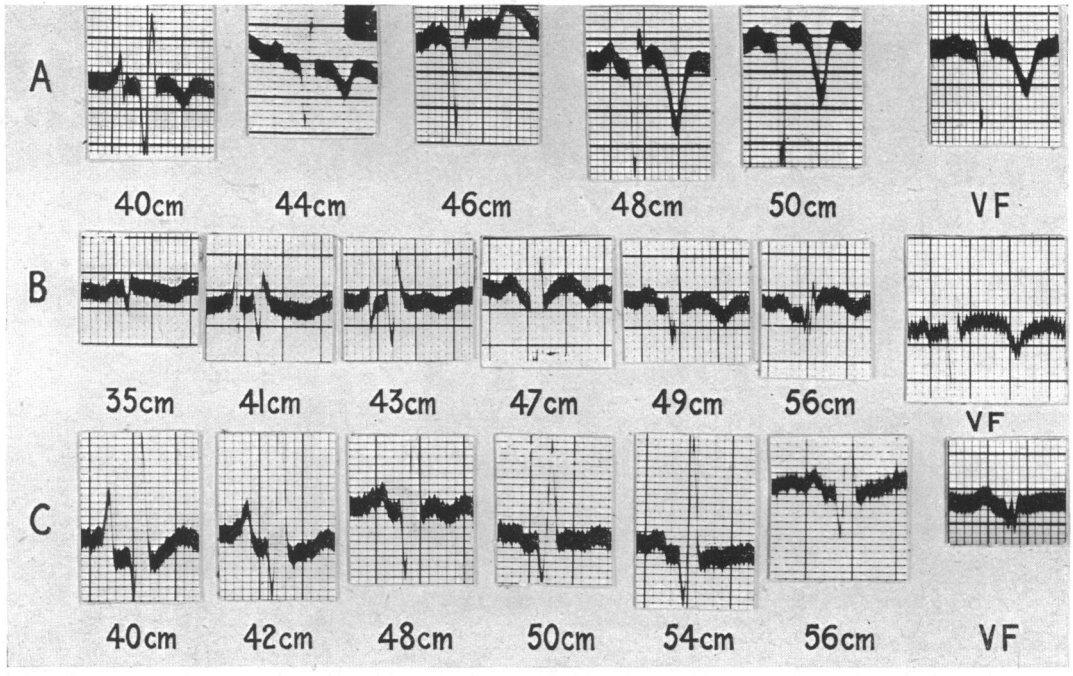

FIG. 12.—*Posterior Infarction.* (A) Case 35 has evidence of recent infarction with deep Q and negative T in lead VF. Similar apperances at 50 and 48 cm. P wave changes do not begin till 40 cm. (B) Case 6 has deep Q and domed T in lead VF. There are similar changes from 55 to 47 cm. P waves alter at 43 cm. (C) Case 52 has evidence of a healed infarct in lead VF with a broad Q, embryonic R, and flat T. A deep Q and flat T are present from 56 to 50 cm. P waves become peaked at 42 cm.

3 cases the deflections at the ventricular level resemble those in V1 and the œsophagus was, therefore, facing the right ventricle. In 2 the complexes were transitional in type, showing that the electrode was opposite the septum. In two of the cases where the œsophagus faced the right ventricle at its lower end, the complexes resembled those of V6, and so faced the epicardial surface of the left ventricle, as high as 30 cm. Both these hearts were grossly hypertrophied, and in hearts with much left ventricular hypertrophy, cavity potentials may never be recorded (Scherlis *et al.*, 1951). In the two cases where the lower end of the œsophagus faced the septum, the changes to the left ventricular surface potentials took place at 47 and 39 cm. (Fig. 13).

Right Bundle Branch Block. In 4 cases the œsophageal deflections at the ventricular level resembled those of lead V5, showing that the œsophagus was facing the left ventricle (Fig. 13). The position of the heart in these cases was either normal or vertical.

DISCUSSION

The œsophageal lead has proved of value in elucidating the nature of auricular arrhythmias when P waves cannot be defined with certainty in the standard or præcordial leads. This has been encountered in complete heart block and in ventricular tachycardia, but it is particularly applicable in some cases of auricular tachycardia, when a rapid and irregular ventricular rate may simulate auricular fibrillation. In these circumstances the œsophageal lead will furnish the diagnosis and record the auricular rate with precision.

The results in auricular flutter and fibrillation are confusing, since the curves are very different. In flutter a sharp narrow intrinsicoid deflection occurs which is usually of high voltage. In fibrilla-

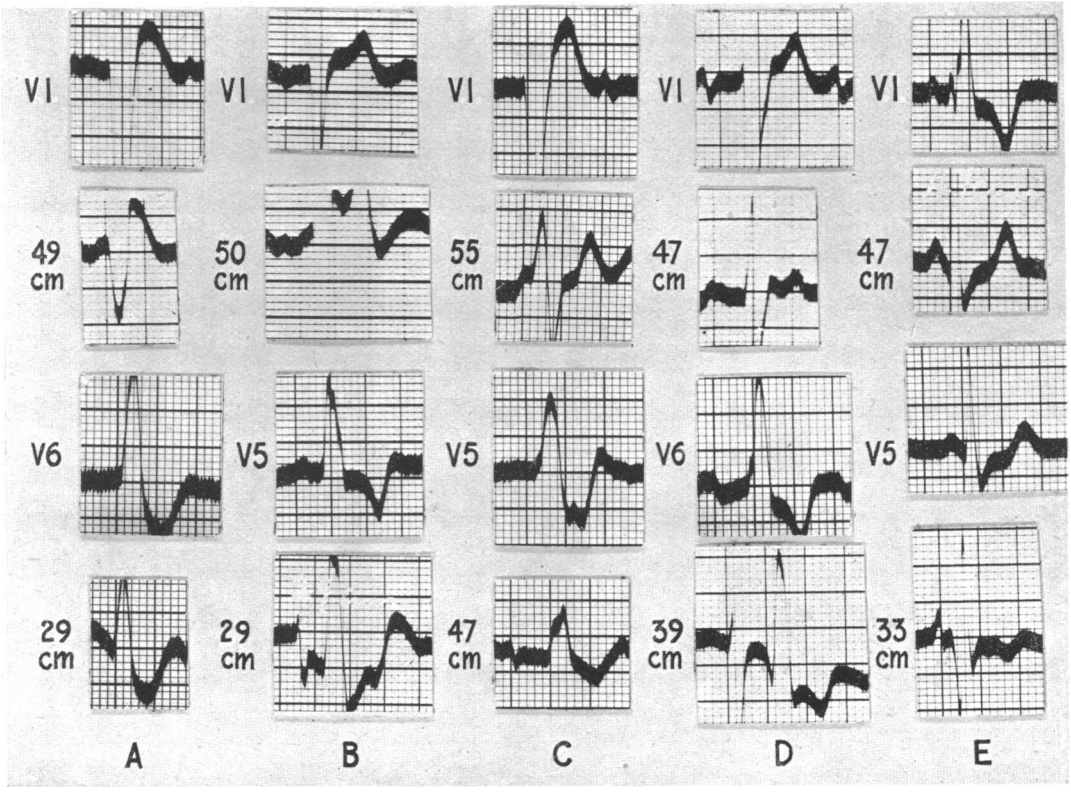

FIG. 13.—(A)–(D). Four cases of left *bundle branch block* and (E) one of right to show which ventricle the œsophagus faced at its lower end. In (A) and (B) Cases 25 and 31 the complexes at 50 cm. resembled V1, and therefore faced the right ventricle. At 29 cm. they resembled V6 and so faced the epicardial surface of the left ventricle. (C) and (D) Cases 1 and 24 show transitional complexes at 55 cm. and 47 cm. respectively, and so faced the septum. At 47 and 39 cm. respectively they resembled V6. (E) In Case 26 with right branch block the complexes at 47 cm. resembled V5, and so faced the left ventricle.

tion there are usually only fibrillary waves, and when large deflections do occur they are broader than normal. In flutter there was always one level at which the auricular diastolic period was isoelectric, although above and below that level there was an ascending base line as the intrinsicoid deflection merged gradually into the flutter peaks. In fibrillation a return to the base line was never seen, and the curves exhibited the usual waxing and waning in voltage. Brown (1936) attempted to explain the difference between the two arrhythmias by suggesting that flutter was due to a single circus and fibrillation to multiple circuses. Prinzmetal *et al.* (1950), however, has recently brought forward substantial evidence against the presence of a circus movement in either condition. The œsophageal lead is a semidirect lead, comparable with the præcordial leads. The intrinsicoid deflections are the result of potentials gathered from a larger area than in the case of the true intrinsic deflections obtained with electrodes attached to the epicardium. It does not seem likely, if the auricles were being activated seriatim by impulses thrown off from a central revolving circus, that the intrinsicoid deflections would have a normal form. Broader complexes such as are found in fibrillation would be anticipated. Moreover, in all the records, as well as in three of those reported by Enselberg (1951), predominant auricular S waves gave place to predominant auricular R waves as the tube was withdrawn, and it is not easy to see how early intrinsicoid deflections could occur so frequently at the lower auricular levels if activation of the auricle arose from a ring involving the orifice of the great veins at the upper end of the right auricle. The evidence of the œsophageal lead

would appear to favour the theory of a circus movement in fibrillation but a single ectopic focus in flutter.

The study of the ventricular levels of the œsophagus has not yielded any information of value. Posterior infarcts are shown but no better than in lead VF. In high posterior infarcts the area of damage is too close to the transitional zone to make the signs of infarction reliable. In horizontal hearts the œsophagus in its lower reaches faces the right ventricle or the septum with the result that the infarct may show better in lead VF.

Conclusions

The œsophageal lead has only a limited application in electrocardiography.

Except in fibrillation, auricular intrinsicoid deflections can almost always be obtained at the auricular levels. This is useful in auricular tachycardia when there is a doubt about the auricular rate, and when the standard leads simulate fibrillation. P waves have also been difficult to recognize in other leads in some cases of multiple premature auricular systoles, and in complete A-V dissociation.

The œsophageal curves of auricular flutter and fibrillation differ so much as to make it unlikely that they are due to the same mechanism. The evidence favours the view that auricular flutter may be due to a single ectopic focus.

The œsophageal lead at ventricular levels has proved disappointing. In posterior infarction the lead is not superior to lead VF.

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